Modern Concepts of Cardiovascular Disease

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AVIATION AND THE CARDIOVASCULARUS

The general effects of flying on the cardiovascular system are practically nil if we exclude the effects of high altitude and high speed. There is no evidence that continued flying has any deleterious effect on this system until we begin to suffer from the effects of oxygen want or change of direction at high speed. Hence, this article will be devoted to a summary of these phases of aviation medicine.

One of the early effects of oxygen want is noted in an increased pulse rate. The pulse continues to accelerate until the subject's altitude limit is reached. If the individual remains at an altitude below his critical level for any length of time, the pulse slows somewhat but never reaches its ground level. The amount of increase depends on the rate, height and duration of ascent. This is interpreted as an effort to get more oxygen to the tissues by increasing the rate of circulation. It, also according to Schneider, is a sign of probable distress. If oxygen be administered, the pulse rate immediately returns to normal. The tendency for the rate to drop after remaining at a specified altitude is explained by Schneider and Havens as due to a reserve supply of red corpuscles being thrown into the circulation, thereby providing a greater oxygen carrying capacity. When the limit of altitude is reached, some subjects faint, and others become unconscious before fainting.

The blood pressure is affected in various ways. There may be no alteration or a gradual rise until a height 12,000 to 15,000 feet is reached. Then one of the following changes may take place (1) a further increase of the systolic and a gradual decrease of the diastolic pressure; (2) an abrupt increase in the systolic pressure, an increase in the diastolic or in both, followed by an abrupt break in one or both with fainting and circulatory collapse. The increased pulse pressure is also interpreted as a sign of distress. At sustained altitudes of 8,000 to 16,000 feet there is a tendency for the blood pressure to return to normal levels. Again, these developments are cut short by the administration of oxygen.

The effects on the heart itself have been studied by various workers. In the early days it was always assumed that high altitude had a bad effect on the heart and patients with heart disease were advised to avoid high altitudes. It was also noted that a number of cases of fainting and collapse occurred at high or simulated high altitudes.

The earliest work was by Whitney who concluded that there is heart failure following marked cardiac dilatation. He felt that the heart itself was vulnerable to the effects of oxygen deficiency. He thought he could detect distantion of the heart by percussion.

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Two years later LeWald and Turrell undertook a series of experiments in which Roentgen photographs were taken of the heart at every 1,000 feet of altitude. They found that forced inspiration and expiration alone accounted for a difference in the transverse diameter of the heart of at least 3 cm. In most of their cases not only was there no increase in the size of the heart at collapse but the heart was actually smaller due to the blood being largely in the splanchnic area.

Greene and Gilbert did the earliest work on the electrocardiogram. They found that the changes due to oxygen want were slight until the critical stage was reached. Briefly, their findings were: Precrisis changes showed shortening of the P-R interval, decrease in the total time of the R-T interval and a decrease in the amplitude of the T wave. Postcrisis changes showed slowing of the rate, displacement of the pacemaker and interference with normal conduction leading to dissociation. The postcrisis rate dropped sometimes five or more per minute and was associated with the development of auriculo-ventricular rhythm.

All this work of Whitney, LeWald, Greene, and Gilbert was done over twenty years ago.

Recently, Armstrong has said, "The effect of anoxia on the heart muscle is the same as that for any other tissue and the heart will continue to function long after the respiratory centers have become paralyzed. The EKG changes from anoxia consist essentially of a lowering or inversion of the T waves, a depression of the R-T interval, and sometimes a deformity of the QRS."

Kountz and Gruber have shown that anoxia produces characteristic changes in the EKG, the initial change observed by them being a decrease in the amplitude of the T wave followed by inversion if the anoxia is progressive—the latter occurring usually at 30 per cent unsaturation of the arterial blood. Other workers have reported the same findings plus a depression of the RT segment. Graybiel reported slight variations in the P-R interval and QRS complexes.

Benson found that there were no changes in the EKG up to 30,000 feet if oxygen were administered.

Still more recent work on this subject has been done by White. Forty-five subjects were exposed to altitude flights without oxygen. Two series were studied. Twenty-five were exposed to 20,000 feet,

half ascending to that level on one hour, the other half in two hours. The second group of twenty was taken to 15,000 feet and held there for two hours.

In the 20,000 feet group there was a progressive diminution of the height of the T wave in all subjects in all four leads. This decrease in height began as low as 5,000 feet. With the administration of oxygen, there was a restoration to normal height. In the 15,000 feet group there were similar changes but a tendency to return to normal noted. One case developed a shift of the pacemaker. As a rule in the group that ascended more slowly the changes were lessened.

As a result, White recommends the use of oxygen at 7,500 feet as a precaution.

Albers and Koch report the following electrocardiographic changes,—changes in the height of the P wave, lowering of the voltage of the QRS and lowering of the T wave. The ST level was lowered in about 1/5 of cases.

Dill and his associates at the Harvard Fatigue Laboratory state that when benzedrine is administered during exposure to low oxygen tensions, there is a slight increase in pulse rate. Benzedrine has a favorable action in preventing the fall in blood pressure during anoxemia.

The electrocardiographic alterations following the administration of benzedrine are slight. This drug tends to prevent the lowering of the T waves which may occur during exposure to low oxygen tensions.

In 1937 Bishop made a study of the question "Is it safe for the heart patient to fly." The sum and substance was that there were two factors to be considered in the case of the cardiac — one the psychological factor associated with the apprehension of first flight. The other is oxygen want associated with high altitude. Apprehension causes acceleration of the heart rate and in the hypertensive and potential hypertensive an increase in blood pressure.

In a person accustomed to flying and who is philosophic about it, there is no more danger in flying than in going by auto or train except for the effects of altitude, which can be relieved by the administration of oxygen.

Graybiel states that there is no evidence that the normal heart is affected by altitude. In susceptible persons peripheral failure may occur and this is evidenced by circulatory collapse. In persons with diseased hearts there is definite evidence that anginal or even congestiv heart failure may occur, but only in those cases with considerable heart disease who are exposed to the oxygen deficiency of levels of 14,000 or more feet. This, of course, may be prevented by the use of oxygen before such levels are reached. Graybiel concludes that under the conditions of civil or military aviation the normal heart is not damaged, and that only patients with-severe heart disease might be advised against flying on commercial planes.

Smith of the Mayo Clinic found 93 cases of coronary sclerosis in 1831 consecutive clinical records of physicians, bankers, lawyers, clergymen, laborers and farmers. The incidence of coronary sclerosis by groups was as follows: physicians showed a rate of 10.7 per cent, bankers 5.3 per cent, lawyers 4.6 per cent, clergymen 4.6 per cent, laborers 2.6 per cent and farmers 2.6 per cent. The incidence in all classes of mental workers was 6.3 per cent compared to an incidence of 2.6 per cent in all classes of physical workers. Leedham feels that flying as a pilot with its tremendous responsibilities such as property, lives, schedules, mental strain plus irregular hours makes it in a class with the practice of medicine and the degenerative hazards will be about equal.

At extreme altitudes above 30,000 feet the effect of lowered barometric pressure is added to that of oxygen want. This low pressure results in what has been termed aero-embolism and aero-emphysema. Pathologically, it is the same as Caisson disease or the "bends," and air emboli may lodge in the blood vessels of the brain, coronary vessels or other terminal vessels. Prevention is by breathing pure oxygen from the ground up and till return to the ground, and for as long as possible up to one hour before departure. The emboli are nitrogen bubbles released at low pressures and nitrogen can be partially replaced in the system by oxygen. Hence the recommendation to use oxygen as outlined.

There is also an effect on the circulation in acceleration and deceleration as experienced in acrobatic flying, in turns at high speed and in dive-bombing. Those circulatory changes are due to centrifugal force. In positive accelerations there results cerebral anemia due to a pulling of the blood to the splanchnic area and to the extremities, while in negative accelerations, there results cerebral congestion. In the first, the pilot sees "black"—in other words, passes out completely, in the second he sees "red" and, actually, retinal or brain hemorrhages are a possibility.

Positive accelerations are the ones usually dealt with, such as occur in turns at high sped in racing, acrobatic manoeuvers and in pull outs from divebombing. The effects can be lessened by having the pilot wear an abdominal belt which can be inflated exerting sufficient pressure against the abdomen to prevent pulling the blood away from the upper part of the body; by placing the pilot in a crouching or nearly prone posture, so that the force strikes his body transversely instead of from the head to the feet, and by having him yell loudly, fixing his diaphragm thus helping to retain some of the blood in the head.

In summary, therefore, we may state that flying per se has no effect on the cardiovascular system. The effects noted are all due to the low oxygen or low barometric pressure of high altitude and the mechanical effects of high speed, the latter the result of centrifugal force. Oxygen is the preventive and cure of the first group and mechanical devices and methods the preventive and cure of the second.

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